AURICULAR FIBRILLATION: SOME CLINICAL CONSIDERATIONS.

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Auricular fibrillation is a condition in which numerous irregular, very rapid impulses are sent out by the auricles. The auricles do not actually contract, but, on the contrary, remain twitching in diastole. The ventricles, however, respond to a small portion of the impulses and contract in a grossly irregular fashion both as to rate and strength. The condition has been called "delirium cordis" and "pulsus irregularis perpetuus." In the great majority of cases it is easily recognized by auscultation at the apex and palpation of the radial pulse. The absolute arrhythmia heard over the precordium, generally with a rate over 100 (when the patient is first seen), together with a pulse deficit (difference between the apex and radial counts) in a patient with cardiac failure indicate that the auricles are fibrillating. In not more than 10 per cent. of the cases, according to Lewis,[1] will the above proposition not be true and in not more than 10 per cent. will this condition be confused with other arrhythmias, especially numerous extrasystoles. Considerations that will appear later in this paper will help to diminish this error still further.

During the past several years considerable work has been done, using the newer methods of cardiac diagnosis, which has given us a clearer conception of auricular fibrillation. The great importance of this condition is indicated by its frequency, 128 different cases having been observed at the Peter Bent Brigham Hospital in thirty months; while during the same period of time there were admitted 146 cases of such a common disease as lobar pneumonia. These figures become more striking when one considers that the cases of pneumonia are not apt to return, while the cardiac cases keep coming back frequently to the wards, though in the figures given above readmissions are not included. In fact, it is not a rare occurrence to see 10 cases of auricular fibrillation at one time in the wards where there are about eighty beds. Its importance is also indicated by the fact that it is generally a permanent irregularity, and if untreated, seriously embarrasses the circulation. At the same time it can be said there is no group of cardiac cases that responds so well to proper treatment.

For the above reasons a clinical study has been made of all the cases of auricular fibrillation observed at the Peter Bent Brigham Hospital during a period of two and a half years. Only those cases have been considered in which the diagnosis was made by electrocardiographic tracings. The special points that were sought for in analyzing the records were a previous history of rheumatic fever or chorea, the presence of organic mitral disease, and the age of the patient. It became a matter of interest whether any data of diagnostic or prognostic value could be drawn from a large group of cases by studying the relation between the presence or absence of a past history of rheumatic fever, the signs or lack of signs of organic mitral disease, and the age of the patient. Other clinical points, such as the importance of syphilis in auricular fibrillation and the differentiation between organic and relative mitral insufficiency, also have been considered.

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[1] From a lecture given at the Peter Bent Brigham Hospital, October, 1914.

As will be shown below, there seemed to be a fairly intimate relationship between the previous history of rheumatic fever and the subsequent development of organic mitral endocarditis (generally mitral stenosis), with the inception of auricular fibrillation. Lea[2] several years ago, using polygraphic tracings in his study of 69 cases of auricular fibrillation, found rheumatic fever or chorea in 56.6. per cent. There was another definite group of cases in our series in which there was no history of rheumatic fever, no signs of mitral disease, and auricular fibrillation was present; these were all cases of cardiac sclerosis or chronic myocarditis. In the entire series there were 128 cases equally divided between males and females, *i. e.*, 64 males and 64 females. (See Table.) The average age of all the cases was 47.3 years (ranging from seventeen to seventy-five years). The average age of the males and females was practically the same, 48 for the former and 46.6. for the latter.

SYPHILIS AS AN ETIOLOGICAL FACTOR. The role that syphilis plays as an etiological factor in auricular fibrillation was found not to be very important. There were only 11 cases that had a positive Wassermann reaction, *i. e.*, 8.6 per cent., the test having been done as a routine on all cases. When we compare this figure with the general average of all cases admitted to the medical wards, which according to Walker and Haller[3] was 12 per cent., we see that syphilis was less common in cases of auricular fibrillation than in those not having it. The 11 cases consisted of 2 with aneurysms of the aorta, 2 of syphilitic aortitis, and 7 which either had chronic myocarditis or chronic mitral endocarditis, or both. It is noteworthy that a fair number of chronic heart cases when first seen have an enlarged liver resulting from a relative regurgitation through the tricuspid valve and become jaundiced. A positive Wassermann reaction under these circumstances may later prove to be negative if repetition is made after the jaundice has disappeared. This happened in at least one of our cases. It is evident from these figures that syphilis plays an unimportant part in the etiology of auricular fibrillation.

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[2] Some Points in Relation to the Etiology of Auricular Fibrillation, Quart. Jour. Med., 1910-11, iv, 423.

[3] Routine Wassermann Examination of Four Thousand Hospital Patients, Jour. Am. Med. Assn., 1916, lxvi, 488.

Statistical data.	Number of cases.	Per cent.
Auricular fibrillation	128	
Persistent auricular fibrillation	110	85.9
Transient auricular fibrillation	18	14.1
Males	64	50.0
Females	64	50.0
Average age of males (limits seventeen to seventy-five years)	48.0	
Average age of females (limits twenty to seventy-two years)	46.6	
Wassermann positive in the blood	11	8.6
Persistent Auricular Fibrillation:		
Group I. Rheumatic history and mitral endocarditis	39	35.5
Males	15	
Females	24	
Average age of males	35.0	
Average age of females	38.4	
II. No rheumatic history and no mitral endocarditis	34	30.9
Males	23	
Females	11	
Average age of males	57.5	
Average age of females	58.9	
III. Unclassified	37	33.6
Total number of cases in Groups I and III with definite rheumatic history	48	43.6
Autopsy Examinations:		
Hearts examined	261	
Over fifty years of age	107	
Showing chronic mitral endocarditis	2	
Under fifty years of age	154	
Showing chronic mitral endocarditis	21	

TRANSIENT AURICULAR FIBRILLATION. Although auricular fibrillation was at first considered a permanent irregularity, and was called "pulsus irregularis perpetuus," it is now quite definitely known that it need not be so. In fact, K. Fahrenkamp,[4] in a review of 120 cases of auricular fibrillation, all diagnosed by electrocardiograms, found 4 transient cases *i. e.*, 3.5 per cent. It is necessary at this point to decide what is to be called transient and what permanent auricular fibrillation. There are some cases which have definite repeated attacks of palpitation and tachycardia just like the ordinary paroxysmal auricular tachycardia, except that the heart's action is absolutely irregular. Short periods of fibrillation are often observed in cases of auricular flutter after digitalis has been given and before the normal rhythm is resumed. Transient auricular fibrillation also occurs under very varied conditions, *i. e.*, during acute rheumatic fever, postoperative, in hyperthyroidism, etc. In addition it is observed rarely as a terminal event lasting from a few hours to a few days. All cases of this series that have not been observed to become regular in rhythm have been considered as cases of permanent auricular fibrillation, and those that were definitely proved to have returned to a normal rhythm were grouped as transient. Of the 128 cases, 110 were persistent and 18 were transient (14.1 per cent.). This is a much higher figure than has heretofore been noted.

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[4] Vorübergehende komplette Herzunregelmassigkeiten unter dem klinischen Bilde der Arhythmia perpetua mit Boebachtungen über Vaguswirkung. Deutsch. Arch. f. klin. Med., 1914, cxvii, 1.

One has to exercise the greatest care in making the diagnosis of transient fibrillation. If one depended on polygraphic tracings or merely on auscultation frequent errors would be made, as has been shown by Fahrenkamp.[5] Numerous extrasystoles together with a sinus arrhythmia might cause a very irregular pulse, and this would change into a normal rhythm, with rest in bed and simple sedatives, as the extrasystoles disappeared. This, of course, wrongly would have been considered a case of transient fibrillation. Then, again, if an absolutely irregular heart action becomes perfectly regular, as determined by auscultation or palpation of the radial pulse, it is no proof that the auricles have stopped fibrillating. Two cases of this series acted in such a way, and at first were thought to be transient cases. This was supposed to have been brought about by digitalis. Careful examination of the electrocardiograms showed that the auricles were still fibrillating, but that there was complete auriculoventricular heart-block with a regular ventricular rhythm. In both of these cases the ventricular rate was rapid, which did not make one immediately think of an idioventricular rhythm. The possibility of a rapid regular ventricular rate in auricular fibrillation is mentioned by Lewis.[6] The ventricular rate of the first case was 62 and that of the other was 110. Furthermore, the frequency with which transient fibrillation will be noted will in a great measure depend on how carefully the heart rhythm is observed and how frequently electrocardiograms are taken. This might explain the comparatively high incidence of transient cases found in this series.

Of the 18 cases of transient auricular fibrillation here observed, 1 occurred during the lysis of pneumonia, similar to one of the cases described by Krumbhaar,[7] 1 in hyperthyroidism, 1 postoperative (avulsion of the sensory root of the trigeminus for trigeminal neuralgia),[8] 2 during acute rheumatic fever, 4 following auricular flutter, 4 as a result of active digitalis therapy, and 6 were spontaneous or idiopathic cases. Among these latter there were 3 who had chronic myocarditis (2 with and 1 without chronic nephritis similar in type to the cases described by Robinson);[9] 1 had cancer of the esophagus, 1 chronic alcoholism, and 1 chronic endocarditis of the aortic and mitral valves. Cohn[10] has observed transient fibrillation during pneumonia in a large portion of his cases, 12 out of 123, 7 of whom had had no digitalis. Two interesting cases have been observed during the past year who were not patients in the hospital and have therefore not been included in this study. They complained of frequent attacks of palpitation, otherwise they were well. Nothing abnormal could be found on careful examination except the arrhythmia, which was a transient auricular fibrillation. Both happened to be well developed athletic young men. Gossage and Hicks[11] refer to the possibility that patients previously well may suddenly develop auricular fibrillation. It therefore can be stated that the transient form of auricular fibrillation may exist in otherwise normal individuals as well as under many various abnormal conditions.

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[5] Loc. cit.

[6] Clinical Electrocardiography, London, 1913, p. 95.

[7] Transient Auricular Fibrillation, an Electrocardiographic Study, Arch. Int. Med., 1916, xviii, 263.

[8] This last case is counted twice because the fibrillation followed a short period of auricular flutter.

[9] Paroxysmal Auricular Fibrillation, Arch. Int. Med., 1914, xiii, 298.

On careful questioning one is impressed by the frequency with which patients suffering from auricular fibrillation have complained of previous attacks of palpitation. Quite often the symptoms of shortness of breath, pain in the chest, cough, etc., have had a sudden onset, and they were not always precipitated by exertion. In fact, some of the patients complained that the trouble was first noticed when they suddenly awoke during the night and found the heart palpitating. In some cases the history of attacks of palpitation was repeated, with periods free from symptoms in between, and extending over one or more years before the permanent state was assumed. In others there was no repetition of the attacks, and on the contrary the symptoms of circulatory failure seemed to date definitely from the onset of auricular fibrillation. It would seem that many of the cases of permanent fibrillation have gone through transient attacks and that the symptoms complained of by the patient are intimately connected with the occurrence of the irregularity.

PERSISTENT AURICULAR FIBRILLATION. There were 110 cases of persistent fibrillation which arbitrarily have been divided into three groups. The first includes all cases who had a definite history of rheumatic fever or chorea and had signs of chronic organic mitral disease. The second includes all those who gave no history of rheumatic fever or chorea and who were diagnosed[12] as having no organic mitral disease. The third group includes all of the remaining cases. One is immediately impressed by the natural obstacles to such a classification. The histories are taken by different house officers. Occasionally one forgets either to ask or at least to record definitely whether the patient had or had not had acute rheumatic fever or chorea, although it must be said that in all but a few cases there was some statement on this point. Furthermore, some patients knew very little English and probably answered to questions they hardly understood. Finally, one might obtain a history of rheumatic pains or rheumatism. This was generally followed by a short description of the condition which identified it; but unfortunately this description was lacking in some instances and was unsatisfactory in others, so it was decided, in this study, to include in the first two groups only those cases in which a definite statement was made and which had the two qualifications required as mentioned above. If a case had a definite history of rheumatic fever and had no evidence of mitral disease it fell in the third or exceptional group; if the past history was vague, or the clinical diagnosis doubtful, it was included in this last group.

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[10] Irregularities in Pneumonia. Unpublished.

[11] On Auricular Fibrillation, Quart. Jour. Med., 1912-13, vi, 435.

[12] In all these considerations the diagnosis taken was the one which was finally made by Dr. H. A. Christian, physician-in-chief, who saw the larger part of the patients, examined the records, and approved of the diagnoses of all the cases.

It was found by this method that 39 cases (15 males and 24 females), or 35.5. per cent., of the cases of permanent fibrillation, fell in the first group, *i. e.*, having a definite history of one or more attacks of rheumatic fever or chorea and showing signs of chronic mitral endocarditis. Most of these showed definite evidence of mitral stenosis, generally with mitral insufficiency. Two cases were included here with some hesitation, because although there was definite evidence of organic aortic disease the signs overshadowed those of mitral disease. They both had loud murmurs of aortic stenosis and aortic insufficiency which were best heard at the base but were also present at the apex of the heart. The question as to whether there was organic mitral endocarditis was in doubt. The Wassermann blood tests were negative; one of the patients had had four attacks of rheumatic fever and the other one attack. In this respect they stand out as the only rheumatic patients who had auricular fibrillation in whom the question as to whether the mitral valve was involved was uncertain, when there were definite signs of valvular disease. These 2 cases were included in Group I, with the positive cases of mitral disease because the endocarditis was undoubtedly rheumatic in nature. The average age of the females in this group was 38.4, that of the males 35; the former were decidedly more numerous. There were only 2 cases over fifty years of age. One was a man, aged fifty-five years, who had three attacks of rheumatic fever at the ages of sixteen, forty-three, and fifty-two. He had been doing very hard physical work all his life up to the age of fifty-two years. It might well be that the first attack of rheumatic fever did not affect his heart, which would account for the late occurrence of the auricular fibrillation and the mitral disease. The other was a woman, aged fifty-six years, who belonged to a higher class in society than the average hospital patient. She had had rheumatic fever at the ages of four and thirty and chorea at twelve years. During which infection her heart became involved it was impossible to tell. In this connection it is of interest to note that Kemp,[13] following his cases over a considerable period of time, found that 23 per cent. of them went through one or more attacks of acute articular rheumatism without clinical affection of the heart. Therefore it can be said that about one-third of the patients with permanent auricular fibrillation had both a rheumatic history and organic mitral disease, and all but 2 of this group were comparatively young.

Thirty-four, or 31.2 per cent. of the cases, were placed in the second group, *i. e.*, having no history of rheumatic fever or chorea and having no signs of chronic organic mitral disease. They were practically all cases of chronic myocarditis with or without chronic nephritis and hypertension. They frequently showed signs of peripheral arteriosclerosis. Of these there were 23 males and 11 females. The proportion of males to females is just opposite in the two groups, there being a marked preponderance of females in the first and of males in the second group. The average age of the males of the latter group is 57.5 and that of the females 58.9. This is about twenty years more than the average age of the first group. The youngest was forty-four years and the oldest seventy-five. The number of cases that could thus be put into one or the other of these two groups was 73, or 66.4 per cent. of all patients with persistent auricular fibrillation. It seems quite striking that with such narrowly limited criteria as a basis of division so large a percentage of cases could be definitely grouped. It is observed that special emphasis has been put on the fact that the valve lesion must be *chronic* and *organic*. The reason for this is that not infrequently one finds fresh minute vegetations on the mitral or aortic valves, which otherwise appear normal, and which indicate a terminal endocarditis. This, of course, could not have any important influence in the çausation of the auricular fibrillation. Again, a very important consideration is the difference between organic and relative mitral insufficiency. A loud, blowing systolic murmur is frequently heard at the apex, which is transmitted to the axilla and often well heard all over the precordium. This is a common finding in senile hearts or cases of chronic myocarditis, especially if there is an associated hypertension. It signifies a functional insufficiency of the mitral valve due to a temporary or permanent dilatation of the mitral ring, but it is often confused with an organic mitral regurgitation. There does not seem to be any very definite method of differentiating the one from the other, and so one has to weigh whatever evidence there is at hand. It is for this reason that the above considerations and the autopsy findings discussed below are of some importance. If a patient is sixty years old and has no history of rheumatic fever, it is very unlikely that he has any organic mitral disease, even if there may be heard a very loud systolic murmur all over the precordium. On the other hand if the patient is thirty years old and has a definite past history of rheumatic fever or chorea and auricular fibrillation is present a systolic murmur at the apex would indicate that there was an organic mitral endocarditis. In fact, a few of the senile cases of this series were considered by some observers to have organic mitral regurgitation because of loud systolic murmurs, and showed normal valves in postmortem examination. The distinction between relative and organic mitral insufficiency is not of much importance in senile hearts, but is of considerable prognostic significance in the cases of the first group. In this group we found only two patients who were over fifty years of age, and practically all were suffering from cardiac insufficiency. Only 6 of the 39 failed to develop signs of mitral stenosis. A diagnosis of organic mitral regurgitation in a middle-aged or young person signifies that mitral stenosis may very likely develop and that the prognosis as to length of life is generally discouraging.

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[13] On the Prognosis of Acute Articular Rheumatism, with Special Reference to the Cardiac Manifestations, Quart. Jour. Med., 1913-14, vii, 251.

The third group is the most interesting because it contains the doubtful cases, and it is here that some of the above considerations can be of help in the diagnosis. Several of them readily fall into one or the other of the first two groups if studied carefully and when some questionable points are definitely determined. There were 37 cases that did not correspond to either of the first two groups. No satisfactory history was recorded, or there was a diagnosis of organic mitral disease in the absence of a history of rheumatic fever, or there was no organic mitral disease in the presence of a definite history of rheumatic fever. A careful study of these cases trying to apply the two general principles is very enlightening, *i. e.*, that a young patient who has auricular fibrillation and a definite rheumatic history very likely has chronic organic mitral disease; and secondly, that an elderly patient who has auricular fibrillation without a past history of rheumatic fever very probably has no organic mitral disease (although frequently there is a relative functional mitral insufficiency). One might further add that if a patient over fifty years old had definite signs of mitral stenosis and auricular fibrillation, very likely there was a previous history of rheumatic fever or chorea, and that this infection involved the heart late in life.

Of the 37 unclassified cases, in 6 no mention was made as to whether the patient did or did not have rheumatic fever. No further statement need be made concerning them except that there were none over fifty years of age with organic mitral disease. There were 9 who had a definite history of rheumatic fever or chorea, and yet were diagnosed as having chronic myocarditis without organic mitral disease; some of these were definitely diagnosed relative mitral insufficiency. A consideration of the ages of these patients is very significant. One would expect that if these patients did not develop organic mitral endocarditis as a complication of the rheumatic fever (reference has been made above to the observation of Kemp[14] that 23 per cent. of rheumatics do not develop heart disease) they would belong to the group of senile hearts and average over fifty years of age. This was true of all cases except one. This was a woman, aged thirty-two years, who had chorea at the age of ten and rheumatic fever at twenty-four years. She had been complaining of dyspnea on exertion and occasional swelling of the legs, which conditions were gradually becoming worse. On physical examination a blowing systolic and a distinct diastolic murmur were heard by three observers on three different occasions. At no time was it noted that the murmurs were absent. The first heart sound at the apex and the second sound at the pulmonic area were not accentuated. Here it seems that there was sufficient evidence for a diagnosis of organic mitral endocarditis which would bring the case into Group I. Two other cases of this series were of particular interest because the house physician recorded a past history of acute rheumatic fever in them. They were both seen by the author, and one was particularly questioned on this point. His history was that of frequent attacks of acute pain in his big toes, with tenderness, redness, occasionally fever, and sometimes with involvement of the fingers. He was a typical case of gout having a very high uric acid content in his blood. The history of "acute rheumatic fever" was more likely that of acute gout. An autopsy was subsequently performed on this case and the mitral valve was found to be essentially normal. The heart was considerably hypertrophied and showed chronic cardiosclerosis. The other case with a rheumatic history was also one of gout having arteriosclerosis and chronic myocarditis without organic mitral disease. Here again the past history of frequent attacks of acute rheumatic fever might well have been that of acute gout. These 2 cases quite certainly belong to Group II. The remaining 6 of the 9 patients, who had auricular fibrillation, chronic myocarditis without organic mitral disease, and a definite history of rheumatic fever, exemplify the type in whom rheumatic fever did not injure the mitral valve, and its effect on the myocardium is questionable. If it is assumed that these 6 patients completely recovered from the rheumatic fever without injury to the heart, as is very probable, they can be considered as cases of auricular fibrillation coming as a result of chronic myocarditis with arteriosclerosis.

Five patients of this unclassified group had vague past histories; several had mild rheumatic pains or rheumatism, one had growing pains, and one did not know which diseases he had had.

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[14] Loc. cit.

Although nothing definite can be said about them, it happens that the only one over fifty years of age had chronic myocarditis without any valve lesions, while the other four had organic valvular disease. Here again the difference in age incidence in the two types of auricular fibrillation is well shown, the younger ones having valvular disease and the older myocarditis.

There still remained 17 of the exceptional cases which have all been diagnosed chronic mitral endocarditis. These definitely had neither rheumatic fever nor chorea. Three had frequent attacks of acute tonsillitis. In the remaining 14 there was no previous history of any of the acute infections which are regarded as the common causes of endocarditis. No importance was attached to a history of mild sore throats which a large majority of all hospital patients evidently have had. A study of these 17 cases brought out some points worthy of mention. There were 6 over fifty years of age among them. Two were definite cases of mitral stenosis. A third which was diagnosed mitral stenosis returned eighteen months later and showed no evidence of mitral disease. A fourth, which was discharged as mitral insufficiency and questionable mitral stenosis, was examined one year later and only a blowing systolic murmur was heard. A fifth was a woman, aged sixty-seven years, who was thought to have chronic myocarditis, chronic nephritis, hypertension, and mitral stenosis, although at no time did she show a diastolic murmur or thrill. The first sound was sharp and suggested mitral stenosis. Her local physician, who had seen her at home, wrote that he had not found evidence of mitral stenosis. There certainly is some question as to whether she had endocarditis. The sixth was considered a case of chronic myocarditis with mitral insufficiency. No definite statement was made as to whether the insufficiency was relative or organic in nature. As was stated above, there generally is no way of deciding this latter point on physical signs, but as a result of the study of 261 autopsies, which will be discussed below, the age and absence of any rheumatic history point strongly against organic valvular disease. Of the 11 remaining cases who had no history of rheumatic fever under fifty years of age, 10 had definite evidence of mitral stenosis with or without accompanying aortic disease, and they all had mitral insufficiency. It can therefore be said that of all the cases of permanent auricular fibrillation there were only 4 over the age of fifty years who had definite organic mitral disease.

TREATMENT. It is well understood by all clinicians that in the treatment of heart disease one should not interest himself in treating an irregularity. The most important aim is to help the heart muscle. In this connection auricular fibrillation plays a very important role, for the irregularity results essentially in a rapid ventricular rate if the condition is left untreated. This rapid rate tires out the heart, and although the patient at first may have no complaints and show no symptoms of circulatory failure the heart muscle is using up its reserve during this time. The cases which show the transient form of fibrillation do not generally need any treatment, except as is indicated by other manifestations, and in pneumonia when the picture becomes most alarming. Those who have persistent auricular fibrillation should be given digitalis to reduce the rapid heart action. There is no better drug for this purpose than digitalis, or strophanthin when more immediate action is desired.

The various means of treating heart failure were employed in these cases. All patients were put to bed, using back-rests and pillows to aid breathing if necessary. If edema was present they were given Karrel diet, consisting of 800 c.c. of milk a day, with no additional water until the edema was gone or was appreciably reduced. Mechanical removal of fluids from the body cavities was seldom necessary. One measure that is probably not sufficiently used in severe forms of heart failure is bleeding. It was customary with these patients to bleed all who showed evidence of extreme failure of the right side of the heart, *i. e.*, marked cyanosis, pulmonary congestion, enlarged tender pulsating liver, edema, ascites, and dilatation of the right side of the heart. Frequently, with most gratifying results, 400 c.c. to 800 c.c. were removed from the median basilic vein, the relief being immediate. This would help considerably in the course of the treatment before the other measures could begin to show their effect. Although most patients were immediately given digitalis in some form, those who were not quite so urgent were sometimes allowed to go without medication for the first twelve or twenty-four hours to observe the change brought about by rest in bed. There was no fixed dose of digitalis used, but in general the patients received one gram of the powdered leaves during the first two or three days, depending on the severity of the condition. Digitalis was continued in 0.1 gram doses three times a day until a therapeutic or toxic effect was produced. In the most urgent cases strophanthin (0.0003 to 0.0005 gram doses) was given intravenously, but always only when it was certain that no digitalis had been previously given. Another helpful drug in many of the cases was theocin. Its beneficial effects as a diuretic have recently been reviewed by Christian.[15] He showed that the best results were obtained by theocin in the cardiac cases with edema who had a good kidney function, and that if given after a course of digitalis it was especially efficient as a diuretic. In all cases particular attention was paid to procuring comfortable rest during the night.

It might hardly seem necessary to emphasize the importance of following the apex-rate in conjunction with the rate of the radial pulse in cases of auricular fibrillation. But this practice is by no means general. These two counts frequently show a marked discrepancy at first, and as the condition of the patient improves the two rates tend to coincide. In fact, it frequently happened that as the patient was improving and the apex-rate was falling the radial rate was increasing, *i. e.*, there were more beats strong enough to reach the wrist with fewer impulses arising in the ventricles. A slow radial pulse is of no significance, for it may indicate either that only a portion of the many beats are strong enough to reach the radial artery or that the heart-rate is actually slow. A slow apex-rate is always significant. In most of the cases reviewed here the heart-rate responded quite readily to the above treatment, and hand in hand there resulted an improvement in the clinical condition. There were several in whom the heart-rate was slowed without an accompanying improvement in the symptoms, and in a few neither the rate nor the clinical picture were affected.

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[15] Some Studies of a Diuretic (Theocin), Arch. Int. Med., 1916, xviii, 606.

POSTMORTEM OBSERVATIONS. All the autopsy reports of the Peter Bent Brigham Hospital for about the first three years were analyzed with special reference to the age of the patient and the condition of the heart, particularly of the mitral valve. Two hundred and sixty-one subjects in whom postmortem examinations of the heart were made (some examinations were restricted to the head or abdomen) were included in this study. Of these there were 107 over the age of fifty, or 41 per cent., and only 2 had definite chronic mitral endocarditis. One was a man of sixty years who had rheumatic fever at the ages of sixteen and fifty-five years, and at autopsy the mitral valve showed slight thickening, a ragged border of the valve flaps, and some contraction of the chordæ tendineæ. The other was a man of sixty-seven years who had rheumatic fever at the ages of forty-two and sixty years. A definite fish-mouth mitral valve was found at autopsy, although the main trouble was alcoholic cirrhosis of the liver with marked ascites. It must be stated at this point that the mitral valve is not infrequently found slightly thickened in old people with arteriosclerosis, and this is generally most prominent in the aortic cusp of the mitral valve. This to the pathologist is an indication of a degenerative and sclerotic process and not of a real inflammatory chronic endocarditis. A further postmortem finding that is met with occasionally, is small fresh vegetations on the valves which are otherwise normal. They are regarded as terminal endocarditis and seem to occur under various conditions. They do not represent any evidence of a chronic endocarditis.[16] There were 154 cases under the age of fifty years of whom 21 had definite chronic organic mitral endocarditis. This is 8 per cent. of all the autopsies analyzed and 13.6 per cent. of those under the age of fifty. In striking contrast is the fact that only 1.9 per cent. of the patients dying at an older age showed organic mitral endocarditis pathologically.

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[16] These two considerations are generally accepted according to Dr. E. W. Goodpasture, resident pathologist at the Peter Bent Brigham Hospital.

CONCLUSIONS. From a study of 128 consecutive cases of auricular fibrillation diagnosed by electrocardiograms, and of 261 consecutive autopsies the following general conclusions may be drawn.

1. Auricular fibrillation is a very common condition in a general hospital being met with almost as frequently as lobar pneumonia.
2. Syphilis plays an unimportant role in the etiology of auricular fibrillation.
3. The transient form of auricular fibrillation is much more common than is ordinarily believed. Careful observations and electrocardiographic tracings are necessary in order to avoid missing the transient cases.
4. About one-third of the cases of persistent auricular fibrillation have had one or more attacks of rheumtic fever or chorea and show clinical signs of chronic organic mitral endocarditis. The average age of these patients is about thirty-seven years. About one-third of the cases have not had rheumatic fever or chorea and show no signs of chronic organic mitral endocarditis. The average age of these patients is about fifty-eight years. About one-third of the cases fall into neither Group I or II.
5. Chronic organic mitral endocarditis is quite infrequently found in patients over fifty years of age either in the living as a result of physical examination or in the dead by postmortem examination.
6. Most patients with organic mitral disease develop mitral stenosis and only a small number reach the age of fifty years.
7. The application of these principles in the properly selected cases will aid in the diagnosis of cardiac conditions, giving particular help in the differentiation of relative and organic mitral insufficiency.

TYPHOID MENINGITIS: WITH REPORT OF A CASE.

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THE cerebrospinal and meningeal manifestations of human infection with typhoid bacillus have an abruptness of onset, a distinctiveness of symptoms, and a prognostic value which demand